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Prognostic Value of Modified Glasgow Prognostic Score in Acute Decompensated Heart Failure with Reduced Ejection Fraction

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ABSTRACT

Objective: Acute decompensated heart failure (ADHF) significantly contributes to hospital admissions and is associated with high morbidity and mortality. This study aimed to assess whether the Modified Glasgow Prognostic Score (mGPS) could predict long-term mortality in patients with heart failure with reduced ejection fraction (HFrEF) admitted for ADHF.

Materials and Methods: HFrEF patients admitted for ADHF between January 2022 and March 2023 were retrospectively analyzed. Patients were stratified by mGPS into three groups: low risk (C-reactive protein [CRP] ≤10 mg/L, albumin ≥35 g/L), moderate risk (CRP >10 mg/L, albumin ≥35 g/L), and high risk (CRP >10 mg/L, albumin <35 g/L). The primary endpoint was all-cause mortality, determined from hospital records and telephone follow-up, and analyzed using Kaplan-Meier (KM) survival analysis.

Results: Of 238 patients (predominantly male, 70%), those with high mGPS were significantly older and had a higher prevalence of hypertension, diabetes, and ischemic etiology. They also exhibited higher creatinine and white blood cell (WBC) counts, along with lower hemoglobin and albumin levels. Mortality was significantly higher in this group. Multiple Cox regression analysis identified high mGPS scores, older age, reduced left ventricular ejection fraction (LVEF), lower hemoglobin, and hypoalbuminemia as independent predictors of mortality. KM analysis demonstrated significantly reduced survival among patients with elevated mGPS.

Conclusion: The mGPS, which incorporates both inflammatory and nutritional parameters, effectively predicts long-term mortality risk in HFrEF patients hospitalized for ADHF. Routine use of mGPS in clinical practice may improve patient stratification, guide therapeutic decisions, and potentially enhance patient outcomes.

Keywords: Acute decompensated heart failure (ADHF), heart failure with reduced ejection fraction (HFrEF), Modified Glasgow Prognostic Score (mGPS), mortality, N-terminal pro-B-type natriuretic peptide (NT-proBNP).

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INTRODUCTION

Acute decompensated heart failure (ADHF) is defined as a clinical condition requiring hospitalization due to the abrupt or gradual onset of heart failure (HF) symptoms that necessitate immediate medical intervention. It is a leading cause of hospitalizations, particularly among individuals aged 65 years and older, and is associated with high mortality and frequent readmissions.[1] Nearly half of patients diagnosed with ADHF present with precipitating factors such as acute coronary syndrome, arrhythmias (particularly atrial fibrillation [AF]), uncontrolled hypertension (HT), infections, medication noncompliance, and dietary indiscretions.[2] Recent studies emphasize not only the role of cardiovascular conditions such as HT and coronary artery disease but also that of non-cardiovascular comorbidities, including diabetes, chronic renal failure, sleep apnea, and iron deficiency, in individuals with ADHF. These factors pose a significant risk for adverse outcomes and highlight the need for comprehensive prognostic assessment upon admission.[3]

Inflammation is recognized as a key pathophysiological factor in both acute and chronic HF.[4] It is intimately linked to disease progression, comorbidities, and negative outcomes throughout the chronic phase, irrespective of conventional metrics such as left ventricular ejection fraction (LVEF) or New York Heart Association (NYHA) class.[5] Recent studies indicate that C-reactive protein (CRP) can predict long-term all-cause mortality among individuals with ADHF.[6] Furthermore, research has shown that malnutrition correlates with frequent hospitalizations and elevated mortality rates in individuals with ADHF.[7,8] The Glasgow Prognostic Score (GPS) is a grading system that reflects both inflammatory and nutritional status by assessing CRP and serum albumin concentrations. The GPS is currently regarded as the most validated inflammation-based prognostic tool in cancer patients and has also proven effective in predicting prognosis in idiopathic pulmonary fibrosis, inflammatory bowel disease, acute coronary syndrome, and both acute and chronic HF.[9–13] The mGPS, an updated version of GPS, provides a more thorough assessment of systemic inflammatory response. The updated system places greater emphasis on elevated CRP levels in the presence of hypoalbuminemia. Notably, patients with hypoalbuminemia but normal CRP levels are classified as low risk by the mGPS.[14]

This research presents the first documentation of the prognostic significance of mGPS in forecasting long-term all-cause mortality in individuals with ADHF and heart failure with reduced ejection fraction (HFrEF). The use of hospitalization data for risk classification in ADHF is crucial for enhancing treatment decision-making.

KEY MESSAGES

- The mGPS, which integrates inflammatory and nutritional status, independently and robustly predicts one-year mortality in HFrEF patients hospitalized for ADHF.
- Patients with high mGPS exhibit significantly increased mortality rates, advanced age, and elevated inflammatory markers, underscoring the importance of inflammation and nutritional factors in prognosis.
- Routine clinical application of mGPS could facilitate personalized risk assessment and tailored treatment approaches for managing ADHF.

MATERIALS AND METHODS

The retrospective, single-center analysis encompassed HFrEF patients admitted for ADHF at our tertiary care institution between January 2022 and March 2023. Conducted in accordance with the Declaration of Helsinki guidelines, the study obtained approval from the institutional Ethics Committee (AEŞH-BADEK-2024-613, date: 26.06.2024). Due to its retrospective design, informed consent was not obtained, and no personal data were utilized. Patient identification was performed using the hospital database and the International Classification of Diseases, 10th Revision (ICD-10) diagnostic codes.

Patients with an LVEF below 40% and diagnosed with ADHF within the "warm-wet" hemodynamic profile were included if it was their first episode following the diagnosis of HF. Patients were excluded if they had unrecorded CRP and albumin levels; systemic inflammatory or autoimmune diseases; were receiving anti-inflammatory medication; were classified as Child class B or C liver failure; had a history of malignancy, recent infections, or recurrent decompensation; or required inotropic support due to a "cold" hemodynamic profile. The diagnoses of ADHF and HFrEF followed the European Society of Cardiology standards for HF.[1]

The patients' demographic data, medical history, medications, laboratory values (obtained during the initial emergency department admission), and echocardiographic features were accurately recorded. HT was defined as elevated systolic blood pressure (SBP) ≥140 mmHg and/or diastolic blood pressure (DBP) ≥90 mmHg, or the continuous use of antihypertensive medication. Diabetes mellitus (DM) was defined as fasting blood glucose >126 mg/dL, hemoglobin A1c ≥6.5%, or the use of hypoglycemic medication. AF was diagnosed based on clinical evaluation, electrocardiogram findings, or the use of oral anticoagulants. The ischemic or non-ischemic etiology of

HF was determined through a review of clinical history and coronary angiography results. The presence of implanted cardioverter-defibrillator (ICD) or cardiac resynchronization therapy (CRT) devices was evaluated by reviewing medical records and confirmed by device interrogation when available.

Baseline samples collected upon initial presentation to the emergency department were used to measure hemoglobin, white blood cell (WBC) count, sodium, potassium, creatinine, glucose, albumin, CRP, and N-terminal pro-brain natriuretic peptide (NT-proBNP). CRP levels were assessed using a high-sensitivity immunoturbidimetric assay, albumin levels with the bromocresol green (BCG) colorimetric method, and NT-proBNP levels via an electrochemiluminescence immunoassay (ECLIA), following standardized laboratory protocols. The patient's CRP and albumin measurements were recorded, and the modified Glasgow Prognostic Score (mGPS) was calculated accordingly. In the mGPS evaluation, Group 0 included patients with CRP ≤10 mg/L and albumin ≥35 g/L; Group 1 included those with CRP >10 mg/L and albumin ≥35 g/L; and Group 2 comprised those with CRP >10 mg/L and albumin <35 g/L. Patients in Group 0 were categorized as low risk, whereas patients in Groups 1 and 2 were designated as high risk.

Transthoracic echocardiography was performed using the GE Vivid E95 ultrasound system, equipped with a 2D M5Sc-D broadband transducer (GE Vingmed Ultrasound, Horten, Norway), in accordance with established echocardiographic protocols. The examination began with M-mode imaging from the parasternal long-axis view to measure aortic and left atrial diameters as well as end-diastolic dimensions. The biplane Simpson's method was employed to quantify LVEF, ensuring precise evaluation of systolic function. The transmitral flow pattern (E, A, E/A) was determined by positioning the pulse-wave Doppler at the mitral valve tips. Measurement of early diastolic mitral annular velocity (e') was performed via tissue Doppler imaging, followed by calculation of the E/e' ratio to evaluate left ventricular filling pressures. Assessment of tricuspid annular plane systolic excursion (TAPSE) was performed via M-mode imaging to determine the longitudinal systolic function of the right ventricle. Estimation of systolic pulmonary artery pressure (sPAP) was achieved through continuous-wave Doppler, in accordance with the modified Bernoulli equation. All echocardiographic evaluations were performed by a seasoned cardiologist, while imaging analysis were undertaken independently by a second investigator to mitigate observer bias. The echocardiography technician was blinded to clinical information and study group allocations to ensure objective and impartial readings.

The trial's primary endpoint was all-cause mortality. Cardiac-related mortality included deaths caused by HF, sudden cardiac death, and myocardial infarction (MI). In-hospital outcomes were examined using hospital records, while post-discharge outcomes were assessed through follow-up telephone interviews when direct hospital data were unavailable. The mean follow-up duration was 25 months.

Statistical Analysis

Statistical analyses were performed using SPSS software, version 22.0 (IBM Corp., Armonk, NY, USA). Categorical data are presented as counts with corresponding percentages. For continuous variables, normally distributed data are expressed as mean±standard deviation (SD), whereas non-normally distributed data are summarized as median with range (min–max). The Kolmogorov-Smirnov test was used to assess data normality across groups. Based on the distribution, Student's t-test was applied to continuous variables with normal distribution, whereas the Mann-Whitney U test was applied to those not normally distributed. Associations between categorical variables were analyzed using the Chi-square test. Predictors of all-cause mortality were identified through Cox proportional hazards regression. Kaplan-Meier (KM) survival curves illustrated event probability over time. A two-tailed p-value below 0.05 was considered statistically significant for all analyses.

RESULTS

The study included 238 patients, categorized into two groups according to their mGPS: Low mGPS (=0, n=114) and High mGPS (≥1, n=124). As shown in Table 1, patients in the High mGPS group had a significantly higher mean age compared to those in the Low mGPS group (67.46±11.15 vs. 51.50±11.47 years, p<0.001). Although male patients accounted for a higher percentage in the High mGPS group (66% vs. 75%), this difference did not reach statistical significance (p=0.149). The groups did not differ significantly in body mass index (BMI) (25.2±3.8 vs. 26.5±2.97 kg/m^2, p=0.619), SBP (129±15.9 vs. 126±19.1 mmHg, p=0.142), DBP (74.33±12.25 vs. 77.42±13.15 mmHg, p=0.165), or heart rate (94.35±11 vs. 93.50±12 bpm, p=0.858). Similarly, LVEF did not differ significantly between the groups (32.34±6.33 vs. 29.74±8.40%, p=0.123).

Echocardiographic parameters, including mitral E-wave velocity (94.99±12.83 vs. 92.86±15.1 cm/s, p=0.213), mitral A-wave velocity (61.82±10.67 vs. 62.10±12.1 cm/s, p=0.126), E/A ratio (1.55±0.60 vs. 1.52±0.47, p=0.734), and E/E' ratio (9.15±1.33 vs. 8.17±1.27, p=0.413), were similar between groups. Parameters related to right ventricular function, such as TAPSE (1.88±0.31 vs. 1.85±0.32 cm, p=0.714) and sPAP (35±10 vs. 32±12 mmHg, p=0.214), also showed no significant differences.

Regarding comorbidities, hypertension was more prevalent in the High mGPS group (90% vs. 32%), though this difference

Table 1. Clinical characteristics and one-year outcomes of the patients

	Low mGPS=0 (n=114)	High mGPS>1 (n=124)	p
Age (years)	51.50±11.47	67.46±11.15	<0.001
Male, n (%)	86 (75%)	83 (66%)	0.149
BMI, kg/cm^2	26.5±2.97	25.2±3.2	0.612
SBP, mmHg	126±19.81	123.69±18.11	0.790
DBP, mmHg	77.42±13.15	74.33±12.25	0.688
Heart rate, bpm	93.50±12	94.35±11	0.858
LVEF, (%)	29.74±8.40	32.34±6.33	0.123
Mitral E, cm/s	92.86±15.1	94.99±12.83	0.213
Mitral A, cm/s	62.07±10.1	61.82±10.67	0.126
E/A	1.64±0.32	1.55±0.13	0.734
E/E'	8.17±1.27	9.15±1.33	0.413
TAPSE, cm	1.92±0.32	1.88±0.13	0.284
sPAP, mmHg	32±0.32	35±0.13	0.214
HT, n (%)	37 (32%)	112 (90%)	<0.001
DM, n (%)	32 (28%)	56 (45%)	0.006
Ischemic etiology, n (%)	38 (33%)	69 (55%)	0.009
AF, n (%)	52 (45%)	56 (45%)	0.312
Cardiac devices (ICD or CRT), n (%)	63 (55%)	71 (57%)	0.757
Glucose, (mg/dL)	102 (69–373)	109 (60–368)	0.092
Creatinine, (mg/dL)	1.00 (0.57–8.00)	1.29 (0.6–5.95)	<0.001
Hemoglobin, (g/dL)	12.85±1.86	12.1±1.97	0.003
WBC, (g/dL)	8.1(3.4–20.3)	9.1 (3.1–19.8)	0.008
Potassium, (mmol/L)	4.36±0.53	4.29±0.59	0.323
Sodium, (mmol/L)	137 (118–147)	137 (122–156)	0.995
NT-proBNP, (pg/mL)	10,924 (5,742–16,832)	12,123 (4,376–18,924)	0.009
Beta blocker, n (%)	113 (98%)	123 (99%)	0.952
ACE inhibitor/ARB, n (%)	103 (92%)	90 (72%)	<0.001
MRA, n (%)	73 (64%)	66 (53%)	0.091
Mortality, n (%)	27 (23%)	65 (52%)	<0.001

Data are presented as mean±standard deviation, median (interquartile range, IQR), or number (%). p<0.05 indicates statistical significance. Abbreviations: ACEi/ARB: Angiotensin-converting enzyme inhibitor/Angiotensin II receptor blockers; AF: Atrial fibrillation; Beta blocker: Beta-blocker therapy; BMI: Body mass index; Creatinine: Serum creatinine levels; DBP: Diastolic blood pressure; DM: Diabetes mellitus; E/A: Ratio of early to late diastolic mitral inflow velocities, E/E': Ratio of early mitral inflow velocity to early diastolic mitral annular velocity; Glucose: Serum glucose levels; Hemoglobin: Hemoglobin levels; HT: Hypertension; ICD/CRT: Implantable cardioverter defibrillator/Cardiac resynchronization therapy; Ischemic etiology: Ischemic heart failure etiology; LVEF: Left ventricular ejection fraction; Mitral A: Late diastolic mitral inflow velocity; Mitral E: Early diastolic mitral inflow velocity; MRA: Mineralocorticoid receptor antagonist; NT-proBNP: N-terminal pro-brain natriuretic peptide; Potassium: Serum potassium levels; sPAP: Systolic pulmonary artery pressure; SBP: Systolic blood pressure; Sodium: Serum sodium levels; TAPSE: Tricuspid annular plane systolic excursion; WBC: White blood cells.

did not reach statistical significance (p=0.092). DM was significantly more prevalent in the High mGPS group (45% vs. 29%, p=0.006). The groups did not differ significantly in ischemic etiology (54% vs. 33%, p=0.003), AF (28% vs. 24%, p=0.430), or the presence of cardiac devices such as implantable ICD or CRT devices (57% vs. 55%, p=0.757).

Regarding laboratory parameters, the High mGPS group had significantly higher creatinine levels (1.20 [0.59–5.05] vs. 1.00 [0.57–0.80] mg/dL, p<0.001) and lower hemoglobin levels (12.12±1.97 vs. 12.85±1.86 g/dL, p=0.003). WBC count was also significantly higher in the High mGPS group (9.1 [3.19–15.9] vs. 8.1 [3.4–20.3] g/dL, p=0.008). Potassium (4.29±0.59 vs.

Table 2. Regression analysis of factors associated with one-year mortality

	Univariable			Multivariable		
	HR	95% CI	p	HR	95% CI	p
HT, n (%)	1.466	1.291–0.746	0.001	0.795	0.422–1.498	0.478
DM, n (%)	1.120	0.732–1.714	0.601			
Hemoglobin (g/dL)	0.857	0.766–0.958	0.007	0.886	0.793–0.990	0.033
WBC (g/dL)	0.983	0.897–1.079	0.724			
LVEF	0.945	0.920–0.969	<0.001	0.915	0.885–0.945	<0.001
mGPS	3.606	2.225–5.844	<0.001	3.739	1.861–7.511	<0.001
Age (years)	1.041	1.024–1.059	<0.001	1.026	1.004–1.047	0.018
Glucose (mg/dL)	1.002	0.998–1.006	0.998			
Sodium (mmol/L)	0.994	0.936–1.044	0.813			
Ischemic etiology, n (%)	1.545	1.025–2.329	0.038	1.186	0.756–1.861	0.457
Creatinine (mg/dL)	1.002	0.826–1.215	0.986			
ACE inhibitor/ARB, n (%)	0.666	0.405–1.094	0.109			
NT-proBNP	1.000	1.000–1.000	0.029	1.000	1.000–1.000	0.267

p<0.05 indicates statistical significance. ACEI/ARB: Angiotensin-converting enzyme inhibitor/Angiotensin II receptor blocker; CI: Confidence interval; DM: Diabetes mellitus; HR: Hazard ratio; HT: Hypertension; LVEF: Left ventricular ejection fraction; mGPS: Modified Glasgow Prognostic Score; MRA: Mineralocorticoid receptor antagonist; NT-proBNP: N-terminal pro-brain natriuretic peptide.

4.36±0.53 mmol/L, p=0.096) and sodium levels (137 [122–156] vs. 137 [118–147] mmol/L, p=0.995) were similar between groups. NT-proBNP levels were higher in the High mGPS group, but the difference was not statistically significant (12,123 [4.376–18.924] vs. 10,924 [5.742–16.832] pg/mL, p=0.996).

The groups did not differ significantly in the use of beta-blockers (99% vs. 93%, p=0.952), angiotensin-converting enzyme inhibitors (ACEi) or angiotensin receptor blockers (ARB) (79% vs. 64%, p=0.093), or mineralocorticoid receptor antagonists (MRA) (53% vs. 64%, p=0.063).

Importantly, the High mGPS group exhibited a significantly higher one-year mortality rate compared to the Low mGPS group (53% vs. 23%, p<0.001).

As shown in Table 2, univariable regression analysis revealed that hemoglobin levels (hazard ratio [HR]: 0.857, 95% confidence interval [CI]: 0.766–0.958, p=0.007), LVEF (HR: 0.945, 95% CI: 0.920–0.969, p<0.001), mGPS (HR: 3.606, 95% CI: 2.225–5.844, p<0.001), and age (HR: 1.041, 95% CI: 1.024–1.059, p<0.001) were significantly associated with increased one-year mortality. Additionally, ischemic etiology (HR: 1.545, 95% CI: 1.025–2.329, p=0.038) and NT-proBNP (HR: 1.000, 95% CI: 1.000–1.000, p=0.029) showed significant associations with mortality.

In multivariable Cox regression analysis, mGPS was a robust independent predictor of one-year mortality (HR: 3.739, 95% CI: 1.861–7.511, p<0.001). LVEF (HR: 0.915, 95% CI: 0.885–0.945, p<0.001), hemoglobin levels (HR: 0.886, 95% CI: 0.793–0.990, p=0.033), and age (HR: 1.026, 95% CI: 1.004–1.047, p=0.018) also retained statistical significance. However, hypertension (p=0.478), ischemic etiology (p=0.457), and NT-proBNP (p=0.267) were not independently associated with one-year mortality in the adjusted model.

These findings highlight the prognostic value of mGPS, along with traditional markers such as LVEF, hemoglobin, and age, in predicting long-term outcomes in patients with HFrEF hospitalized for ADHF.

KM curves (Fig. 1) demonstrate a clear survival difference between the two mGPS groups. Patients with High mGPS exhibited lower survival rates throughout the study period, indicating an association between higher mGPS scores and worse prognosis.

DISCUSSION

This study represents the first investigation into the impact of the mGPS, a simple yet effective risk stratification tool, on long-term mortality among HFrEF patients admitted for ADHF. The findings demonstrate that mGPS, as an indicator of both nutritional and inflammatory status, serves as a reliable predictor of long-term prognosis. Notably, an elevated mGPS is associated with older age and specific cardiometabolic parameters, indicating an increased risk of all-cause mortality within this patient population. These results align with existing literature, underscoring the pivotal

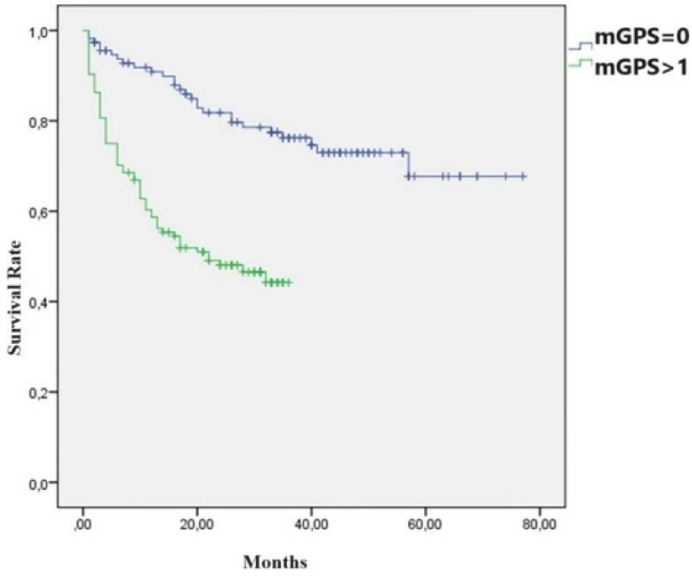

Figure 1. Illustrates that patients with high modified Glasgow Prognostic Score (mGPS) have a higher mortality rate than those with a low score.

role of inflammation and nutritional status in shaping the clinical outcomes of HF patients. By incorporating CRP and albumin levels, mGPS appears to be an effective marker for stratifying risk among patients with ADHF.

An acute deterioration in cardiac function, often involving worsening left ventricular (LV) diastolic function, elevated LV filling pressures, and subsequent pulmonary congestion, can lead to ADHF.[15] Globally, in-hospital mortality rates for ADHF are approximately 4%, rising to nearly 10% at 60–90 days post-discharge and 25–30% within the first year.[16] Among patients with HFrEF presenting with ADHF, mortality risk further increases as ejection fraction (EF) declines. The progression of HF toward mortality is primarily driven by reduced cardiac output, severe hypoperfusion, malignant arrhythmias, advanced pulmonary edema, prolonged neurohormonal activation, and heightened inflammation and oxidative stress. As EF decreases, the inflammatory response intensifies, contributing to HF progression. Reduced EF leads to impaired tissue perfusion and oxygen deprivation in cardiac tissues, triggering neurohormonal activation—particularly the renin-angiotensin-aldosterone system (RAAS) and the sympathetic nervous system—alongside oxidative stress and microvascular dysfunction. These pathological processes collectively stimulate the release of inflammatory mediators, including key cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-1 (IL-1), and interleukin-6 (IL-6).[17]

TNF-α was the earliest cytokine identified to show a significant rise in the serum of patients with HF and is implicated in myocardial apoptosis and necrosis, ultimately contributing to adverse ventricular remodeling.[18] Elevated TNF-α levels are frequently associated with impaired systolic function and poorer long-term survival outcomes. Similarly, IL-6, a cytokine predominantly synthesized by monocytes, exhibits a direct association with HF severity while demonstrating an inverse correlation with LVEF and overall survival.[19] IL-6 stimulates hepatic synthesis of acute-phase reactants, including CRP and fibrinogen, while concurrently downregulating albumin transcription. This process increases the demand for specific amino acids, potentially contributing to hypoalbuminemia.[20] The Framingham study reported that increased CRP levels were linked to a 2.8-fold greater risk of developing congestive HF.[21] In chronic HF, elevated CRP levels are well recognized, with serum concentrations correlating with functional limitations and prognosis. However, CRP levels do not correlate with the severity of left ventricular dysfunction as assessed by ejection fraction.[22,23]

Hypoalbuminemia commonly occurs in patients with HFrEF, with its prevalence increasing with advancing age and disease progression. It is independently associated with a higher mortality risk.[24] The primary etiologies of hypoalbuminemia include malnutrition, systemic inflammation, and cachexia, although other contributing factors such as hemodilution, liver failure, and nephrotic syndrome may also contribute. In the context of HF, hypoalbuminemia is thought to facilitate transcapillary escape, despite the lack of a direct correlation between low albumin levels and impaired cardiac function. Serum albumin significantly influences a patient's overall physiological state, potentially modifying the clinical presentation of HF. Although albumin is considered a modifiable risk factor in several cardiovascular conditions, it is still unclear whether nutritional interventions or albumin supplementation provide meaningful clinical benefits for HFrEF patients with hypoalbuminemia.[25]

Liver dysfunction is commonly detected in patients with HFrEF, likely due to shared risk factors and complex cardiohepatic interactions. The liver, as the sole site of albumin synthesis, is particularly vulnerable to the hemodynamic disturbances induced by HFrEF, including chronic hypoxia and venous congestion. These factors impair hepatic function and reduce albumin production, leading to elevated liver enzyme levels and worsening hypoalbuminemia. Both conditions are linked to adverse clinical outcomes and pose significant challenges in the management of HFrEF.[26,27]

Elevated CRP levels, indicative of active inflammation, combined with reduced serum albumin, reflect the body's

systemic response to chronic inflammation. Assessing CRP and serum albumin levels together provides broader insight into a patient's inflammatory status than evaluating either marker alone. The mGPS, an inflammation-based prognostic tool that combines CRP and albumin measurements, is easily obtainable, widely accessible, and highly standardized. Currently, mGPS is the most thoroughly validated for use in cancer patients, offering significant prognostic information across various cancer types, regardless of tumor location, and applicable to both resectable and unresectable disease states. Similar to HF, cancer is a systemic disease characterized by an active inflammatory response, with nutritional and functional decline as key pathophysiological features. Notably, increased levels of pro-inflammatory markers in cancer patients correlate with increased cardiovascular indicators, even in the absence of a confirmed cardiac diagnosis. Despite the recognized importance of inflammation in HF, inflammatory scores like mGPS have not been adopted for risk stratification in HFrEF. Moreover, there is a paucity of research examining the prognostic impact of GPS in ADHF patients.

Previous research has established the mGPS as a reliable predictor of prolonged hospitalizations and mortality in CRT patients. It has demonstrated comparable accuracy to other nutritional scores for forecasting in-hospital mortality in elderly patients, independently predicted survival in stable HFrEF patients irrespective of NT-proBNP levels, and predicted all-cause mortality and HF-related hospitalizations in patients with HF with preserved ejection fraction (HFpEF).[14,28–30]

Our findings highlight the value of personalized treatment approaches in HF management. Identifying hemoglobin levels, LVEF, and age as independent predictors of mortality risk suggests that targeted interventions addressing these factors may improve patient outcomes.

Anemia adversely affects cardiac function by reducing the oxygen-carrying capacity of the blood, leading to myocardial hypoxia, impaired cardiac efficiency, and worsening HF. In response, the heart increases cardiac output, thereby elevating myocardial workload, which may contribute to myocardial hypertrophy and ventricular dilatation. This pathophysiological cascade can result in left ventricular dysfunction, exacerbate HF symptoms, and heighten the risk of myocardial ischemia, particularly among individuals with concomitant coronary artery disease (CAD). Consequently, the probability of angina and MI increases. Chronic anemia further accelerates HF progression, is linked to high hospital readmission rates, and correlates with increased mortality.[31–33] Additionally, anemia activates neurohormonal pathways, including the RAAS and the sympathetic nervous system, resulting in vasoconstriction, fluid retention, and increased cardiac workload, all of which exacerbate HF. Given these interconnections, incorporating anemia-related parameters into prognostic models such as the mGPS may contribute to more effective risk stratification and therapeutic optimization in HF patients.

Future research examining mGPS alongside other established prognostic markers may offer a more holistic understanding of the pathophysiology and management of HF. Such investigations could facilitate the development of personalized therapeutic strategies, ultimately improving survival outcomes and quality of life for patients with HF.

Limitations

This investigation is subject to certain limitations that should be considered. First, its retrospective and single-center design inherently restricts the generalizability of the results, potentially reflecting local clinical practices, patient demographics, and healthcare infrastructure rather than broader populations. Second, the relatively small sample size could limit statistical power, potentially explaining why certain parameters, such as NT-proBNP, creatinine levels, or common comorbidities, did not achieve statistical significance in multivariable Cox regression analysis despite their prognostic relevance in previous studies. Additionally, the strict exclusion criteria, particularly the omission of patients with severe inflammatory conditions, a history of malignancy, or "cold" hemodynamic profiles, may limit the applicability of these findings to the broader population of ADHF patients. Another limitation is that the retrospective design prevented a comprehensive assessment of potentially impactful clinical variables such as intravenous diuretic dosing strategies, dietary interventions, and newer HF therapies (e.g., sodium-glucose co-transporter 2 [SGLT2] inhibitors), which might influence inflammation, nutritional status, and clinical outcomes. Furthermore, the single-center and observational nature of the study inherently limits the external validity and generalizability across different ethnic groups, healthcare settings, and patient demographics. Future multicenter, prospective studies with broader inclusion criteria should validate the prognostic value of mGPS in a wider spectrum of HF populations.

CONCLUSION

This study demonstrates that the mGPS reliably predicts long-term mortality among HFrEF patients admitted for ADHF. As an easily applicable prognostic tool integrating inflammatory and nutritional parameters, mGPS can facilitate early identification of high-risk patients and thereby support the implementation of personalized therapeutic strategies. Routine integration of mGPS into clinical practice may improve patient outcomes. Further multicenter, prospective research should validate these findings and clarify the potential clinical benefits of mGPS-guided management strategies across diverse HF populations.

Ethics Committee Approval: The Ankara Etlik Hospital Ethics Committee granted approval for this study (date: 26.06.2024, number: AEŞH-BADEK-2024-613).

Informed Consent: Written informed consent was obtained from patients who participated in this study.

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